Severe sepsis due to *Mycobacterium tuberculosis* following extracorporeal shock-wave lithotripsy

Clin Microbiol Infect 1998; 4: 108–109

Infection occurs rarely following extracorporeal shock-wave lithotripsy (ECL). In most of the cases bacteria that commonly infect the urinary tract are identified. Only two cases of disseminated urinary tuberculosis following extracorporeal lithotripsy have previously been reported [1,2].

Case report

A 70-year-old woman was admitted to the intensive care unit in our department. Severe sepsis had developed very rapidly in the previous 24 h. Seven days before, she had been treated by ECL to disrupt right kidney stones.

Intravenous urography preceding lithotripsy had shown normal kidney size and no abnormality of the urinary tract except moderate dilatation of the right renal pelvis. Three days after the ECL, her temperature had risen to 38.5 °C. Antibiotics (pefloxacin and amoxicillin/clavulanic acid) had been unsuccessful. Urinary and blood cultures remained sterile. Abdominal echography 2 days later showed a homogeneous hepatomegaly and no dilatation of the urinary tract. The persistence of leukocyturia and presence of acid-fast bacilli in urine the same day indicated the diagnosis of urinary tuberculosis.

On admission to the intensive care unit because of severe sepsis, examination showed low-grade fever (38.2 °C), tachypnea (44/min), confusion and jaundice. Blood analysis showed: hemoglobin 117 g/L, leukocytes 5.6×10^6/L and platelets 22×10^6/L; sedimentation rate 8 mm/h; prothrombin time 43%; fibrinogen: 0.2 g/L; lactic acidosis: 3.48 mmol/L; cholestasis (bilirubin level 129 mmol/L, alkaline phosphatase 1122 UI/L, SGPT 669 UI/L, SPOT 480 UI/L). An HIV test was refused by the patient, but the CD4/CD8 lymphocyte ratio was normal.

The chest X-ray showed interstitial changes, but no miliary or tubercular appearance.

Bone marrow and hepatic biopsies revealed hemophagocytosis. Blood, urine and bone marrow cultures were positive for *Mycobacterium tuberculosis* 3 weeks later. Culture was performed on Lowenstein–Jensen and 13 A Bactec (Beckton–Dickinson) media. There was no resistance noted towards rifampicin, isoniazid, streptomycin and ethambutol.

Intravenous antibiotherapy (rifampicin 10 mg/kg/per day, isoniazid 5 mg/kg per day, ethambutol 20mg/kg per day) was initiated. Methylprednisolone (1,5 mg/kg per day) was added for the first 2 weeks of treatment. The improvement in the patient's mental state was already spectacular after 24 h. The abnormalities of coagulation and the cholestasis lessened progressively. The renal function worsened from the third day after starting antituberculous therapy (creatinine level 370 μmol/L, urea 45 mmol/L at day 14). It improved spontaneously 2 weeks later. The antituberculous drugs were maintained at the same dose.

On day 19 sudden abdominal pain led to a laparotomy, revealing a perforated gastric ulcer. After 48 days of hospitalization the patient was discharged in a good general state of health.

The diagnosis of quiescent tuberculous infection is rare in the check-up preceding ECL. In most cases, if there is urinary infection, it will be due to common bacteria [3]. Renal lithiasis is more frequently associated with recurrent bacterial urinary infection. However, urinary tuberculosis is accompanied in 10.5% of cases by renal lithiasis [4]. When diagnosis of latent urinary infection is omitted, the disruption of multiple renal blood vessels by high-dose ECL in the case of an undiagnosed renal infection may cause major bacteremia. Dissemination of bacteria is infrequently responsible for severe sepsis by sudden discharge of a large quantity of microbial antigens and may then cause systemic inflammatory activation. The infectious syndrome may be more severe when urinary infection is associated with perirenal hematoma or obstruction of the urinary tract [3].

In the two other published cases the diagnosis of urinary tuberculosis was even more delayed (10 and 16 days after lithotripsy, respectively). In both cases, sepsis was less severe and the patients improved after specific treatment [1,2]. Septic shock due to *M. tuberculosis* has been described before in immunodeficient HIV-seropositive patients [5,6]. In immunocompetent patients, severe sepsis with renal and hepatic organ failure only occurs when diagnosis is delayed [7,8]. Corticosteroids were proposed here to reduce the proliferation of the inflammatory cells responsible for major tissue lesions in tuberculosis. There is no indication for corticotherapy in severe sepsis or septic shock at this time. It is unlikely that the corticotherapy caused the gastric ulcer rupture 2 weeks later. Even long-term corticotherapy does not seem to influence the occurrence of gastric ulcer [9].

Because it is rare, the diagnosis of tuberculosis following ECL may be delayed. As in our patient, it can lead to severe sepsis with multivisceral failure.

Tuberculosis should be considered when broad-spectrum antibiotherapy is ineffective and no evident cause is found for urinary infection following ECL.

A. Chaumeron[1], *J. Peceny*[1],
O. Rauzy[2], *L. Cuzin*[1],
B. Marchou[1], *J.Ch. Auvergnat*[1]
[1]Department of Infectious Diseases
(Pr Auvergnat), Purpan Hospital,
Toulouse, France;
[2]Internal Medecine (Pr Carles),
Purpan Hospital,
Toulouse, France

Revised version accepted 16 August 1997

References

1. Federmann M, Kley HK. Miliary tuberculosis after extracorporeal shock-wave lithotripsy. N Engl J Med 1990; 323: 1212.
2. Morano Amado LE, Amador Barciela L, Fernandez AR, Sapina Llamas IM, Vazquez Alvarez O, Fernandez Martin J. Extracorporeal shock-wave lithotripsy complicated with miliary tuberculosis. J Urol 1993; 149: 1532–4.
3. Coptcoat MJ, Webb DR, Kellett MJ, et al. The complications of extracorporeal shock-wave lithotripsy: management and prevention. Br J Urol 1986; 58: 578–80.
4. Lenk J, Schubert G, Oesterwitz H, Brien G. Urolithiasis associated with urogenital tuberculosis: clinical and microbiological aspects. Urol Res 1988; 16: 157–9.
5. Ahuja SS, Ahuja SK, Phelps KR, Thelmo W, Hill R. Hemodynamic confirmation of septic shock in disseminated tuberculosis. Crit Care Med 1992; 6(20): 901–3.
6. George S, Papa L, Sheils L, Magnussen CR. Septic shock due to disseminated tuberculosis. Clin Infect Dis 1996; 22: 188–9
7. Godwin JE, Coleman AA, Sahn SA. Miliary tuberculosis presenting as hepatic and renal failure. Chest 1991; 99: 752–4.
8. Hussain W, Mutimer D, Harrison R, Hubscher S, Neuberger J. Fulminant hepatic failure caused by tuberculosis. Gut 1995; 36: 792–4.
9. Lefering R, Neubauer EA. Steroid controversy in sepsis and septic shock: a metaanalysis. Crit Care Med 1995; 23: 1294–303.

Bacteremia due to *Roseomonas* spp.

Clin Microbiol Infect 1998; 4: 109–112

Pink-pigmented, oxidative Gram-negative bacteria have occasionally been isolated from various clinical samples [1]. However, their clinical significance is still frequently debated [2]. We report a case of catheter-related bacteremia due to a pink Gram-negative coccobacillus, *Roseomonas gilardii*, in an immunocompromised patient and we also review the literature in order to delineate the epidemiology and pathogenic role of *Roseomonas* spp. isolated from blood samples.

Case report

A 59-year-old man with M1-type acute myelogenous leukemia developed fever (39°C) while on chemotherapy, 4 months after the implantation of a Hickman catheter. One out of the four blood cultures obtained through a peripheral vein grew pink-pigmented bacteria and the patient was afebrile when discharged after 10 days of intravenous imipenem and tobramycin followed by oral ciprofloxacin for 5 days. The patient was re-admitted 2 weeks later with a new febrile episode (39°C), and a similar microorganism was isolated in two out of three blood cultures. The patient defervesced with intravenous ceftazidime for 13 days followed by oral ciprofloxacin for 10 days and was discharged. A third episode occurred 2 weeks after the second discharge. Lysis–centrifugation blood cultures obtained through both catheter lumens showed colonies too numerous to count. A simultaneous peripheral blood sample treated with lysis–centrifugation showed no growth. The Hickman catheter was removed after 10 days but unfortunately the tip was not sent for culture. The patient was discharged with oral ciprofloxacin and cloxacillin for 7 days. Five months later he has not had further episodes of bacteremia. All pink-pigmented bacteria grown from blood cultures of the three episodes of bacteremia were identified as *Roseomonas gilardii*.

In all cases, pink-pigmented colonies grew after 48 h of incubation at 37°C on Columbia blood agar plates. The Gram stain revealed Gram-negative, non-vacuolated coccoid rods, arranged in pairs. The organism grew on MacConkey agar and weakly on Columbia blood agar at 42°C. Catalase and oxidase tests were positive, although the latter test was delayed for 30 s. The organism was non-fermentative on triple sugar iron agar, and the API 20NE identification system (BioMérieux, Marcy L'Etoile, France) gave positive results for urease production, and oxidation of glucose, arabinose, malate and citrate, while nitrate reduction, glucose fermentation, indole production, arginine dihydrolase, β-glucosidase (esculinase), gelatinase and β-galactosidase tests and oxidation of mannose, mannitol, *N*-acetylglucosamine, maltose, gluconate, caprate, adipate and phenylacetate were negative (code number 0241045). The API computer database identified the microorganism as *Pseudomonas mesophilica*, and citrate oxidation was the only unacceptable test. Because this database does not include the genus *Roseomonas*, further tests were performed to confirm the identification.